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PROCEEDINGS

OF THE

PATHOLOGICAL SOCIETY

OF

PHILADELPHIA

New Series, Vol. VI. January, 1903 No. 3

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Third Annual Exhibition Meeting of the Pathological Society of Philadelphia.—McFarland, Angiosarcoma of the Hypophysis Cerebri without Acromegaly.—Githens, A Fatal Case of Anthracosis, with Autopsy.—Cohen and Riesman, Atrophic Cirrhosis of the Liver.

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Third Annual Exhibition Meeting.

The third annual exhibition meeting of the Pathological Society of Philadelphia was held in the Mütter Museum of the College of Physicians, December 11, 1902. The exhibition was devoted chiefly to gross morbid anatomy, and comprised an unusually large number of interesting and instructive specimens, a list of which is given below:

Dr. B. M. Anspach.

1. Adenomatous proliferation of the cervical appendages of the duct of Gärtner. These appendages are analogous to the seminal vesicles of the male. Two cases have been reported—one by Meyer; the other by Thumin. Specimen is from latter's case.
2. Hypernephric tumor of the broad ligament.
3. Cystic endothelial sarcoma of the ovary.
4. Tubal pregnancy, showing the penetration of the wall by chorion epithelium.
5. Myxoma peritonei.
6. Uterine mucosa, showing either chorion wandering cells or decidual cells in the vessel walls. If the former, an instance of the destructive or penetrating power of these cells. If the latter, perhaps an indication of their origin.

7. Wall of uterus following labor. Showing thrombosed vessels and penetrating chorion epithelium.

Drs. Charles W. Burr and **D. J. McCarthy**.

1. Thrombosis of cerebral veins, with hemorrhagic extravasation.
2. Cerebral thrombosis. Anterior cerebral distribution.
3. Multiple thrombosis of cerebral cortex (extensive arterial disease).
4. Cerebral thrombosis. Sylvian distribution.
5. Thrombosis of Sylvian artery. Branch to ascending parietal convolution.

Dr. W. M. L. Coplin, from the Laboratories of the Jefferson Medical College Hospital.

1. Nephrolithiasis, with slight hydronephrosis.
2. Kidney. Primary cancer of the adrenal, with carcinomatosis of the kidney-bed.
3. Half of kidney, showing posttyphoidal pyelitis.
4. Half of kidney, showing hydronephrosis.
5. Slab of kidney, showing acute suppurative interstitial nephritis of embolic origin. Embolic abscess.
6. Small pyramids of kidney, showing anemic infarcts.
7. Half of kidney, showing chronic interstitial nephritis, with a single, rather large anemic infarct.
8. Half of kidney, chronic interstitial nephritis—contracting kidney.
9. Slab of kidney, chronic parenchymatous nephritis—large white kidney.
10. Half of kidney, showing subacute diffuse nephritis.
11. Half of kidney, showing acute diffuse nephritis, with anemic infarct.

Dr. M. H. Cryer, University of Pennsylvania.

Series of 12 frozen sections illustrating anatomy of the head. Photographs of the same.

Dr. G. G. Davis, University of Pennsylvania.

1. Longitudinal frozen section of the head and trunk.
2. Wrist-joints.

3. Elbow-joint injected with wax.

4. Ligaments of the elbow, orbicular, lateral, and capsular.

5. Shoulder-joint injected with wax.

6. Dislocation of the thumb.

7. Renal calculus—calcium carbonate and ammonium magnesium phosphate.

8. Renal calculus—oxalate of lime (mulberry calculus).

9. Renal calculus—triple phosphates. From the right kidney of a young man.

10. Biliary calculi. From gall-bladder and cystic duct.

11. Dissected preparation showing musculospiral nerve, etc.

12. Brains prepared for teaching fissures, convolutions, ganglia, etc.

13. Subcoracoid dislocation of the shoulder. Showing position of vessels and nerves.

Drs. F. X. Dercum and William G. Spiller.

Sclerosis in upper motor area of brain.

Dr. Charles H. Frazier, from Dr. Frazier's collection of charts used for the purpose of demonstrating the pathology of surgical affections in the course on Surgical Pathology, University of Pennsylvania.

1. Goiter.
2. Scirrhus of breast.
3. Osteosarcoma.
4. Appendicitis.

Dr. F. P. Gay, University of Pennsylvania.

1. Bacillus dysenteriæ. Comparison of agar cultures from various sources and cultural characteristics of the organism on various media.

2. Colonies of Bacillus dysenteriæ on agar-agar.

3. Antidysenteric serum.

4. Dysentery vaccines. Suspended cultures of the organism, killed and preserved with 0.5 per cent. tricresol.

5. Hemorrhagic nephritis in the guinea-pig, one of the evidences of the toxicity of the dysentery vaccine.

6. Bacillus dysenteriæ—stained to show flagella.

7. Experimental eliminative lesions in cecum of rabbit 60 hours after subcutaneous injection of culture of Bacillus dysenteriæ.

8. Bacillus dysenteriæ. Pfeiffer's reaction, showing extracellular bacteriolysis in the peritoneal cavity of the guinea-pig when the organism is introduced, together with the antidysenteric serum.

Drs. W. W. Keen and **William G. Spiller.**

Multiple fibromata of ulnar nerve.

Drs. A. O. J. Kelly and **W. J. C. Bryan,** German Hospital Pathological Institute.

1. Double pyohydronephrosis. Hypertrophy of both ureters. Papilloma of bladder.
2. Acute hemorrhagic pancreatitis, with necrosis. Focal areas of fat necrosis.
3. Blood cast of intestine. Removed postmortem.
4. Acute appendicitis; twenty-four hours' duration.
5. Bilateral interstitial nephritis.
6. Aneurysm of ascending arch of aorta, with recent clot. Aneurysm of upper part of transverse arch, with organized clot. Dilatation of lower part of transverse arch. Multiple atheroma of aorta.
7. Aneurysm of left side of ascending aorta. Dilatation of whole arch. Multiple atheroma of aorta.
8. Gangrenous appendicitis.
9. Pyohydronephrosis.
10. Nephrolithiasis. Pyohydronephrosis.
11. Nephrolithiasis. Pyohydronephrosis.
12. Multiple cysts of both kidneys.

Drs. James Hendrie Lloyd and **D. J. McCarthy.**

Thrombosis of veins of Galen and straight sinus, also of the veins of the cerebellum and bulb.

Dr. W. T. Longcope, Pennsylvania Hospital.

1. Rupture of abdominal aneurysm about right kidney. Atrophy of kidney.
2. Osteosarcoma of humerus. Male, aged seventeen years.
3. Hemorrhagic pancreatitis. Multiple fat necrosis.

4. Periosteal sarcoma of femur. Male, aged twenty-three years.

5. Carcinoma of cervix.

6. Primary carcinoma of gall-bladder. Concretions in gall-bladder.

7. Multiple gummata of liver, acute cholangitis, dilatation of hepatic bile-ducts and common bile-duct. No constriction of common duct.

8. Thrombosis of splenic artery caused by embolus from acute vegetations upon aortic valve. Complete necrosis of spleen.

9. Extreme arterial sclerosis of aorta.

10. Aneurysm of descending arch of aorta. Rupture into esophagus. Male, aged thirty-seven years. Death occurred suddenly in third week of an attack of typhoid fever. Patient raised himself in bed, vomited about one quart of blood, and fell back dead.

11. Acute mitral ulcerative endocarditis. Acute mural endocarditis. Cultures—Micrococcus lanceolatus.

12. Chronic interstitial myocarditis and chronic mural endocarditis. Partial obliteration of left coronary artery.

13. Primary carcinoma of gall-bladder, extension to cystic duct, common bile-duct, and hepatic duct. Occlusion of hepatic duct, dilatation of hepatic duct and bile-ducts of liver. Extension of growth to pancreas, occlusion of duct of Wirsung, atrophy of tail of pancreas, and multiple fat necrosis

14. Anemic infarction of soleus muscle. Thrombosis of femoral, popliteal, and posterior tibial arteries, with occlusion of smaller branches supplying soleus muscle.

15. Anemic infarction of spleen. Typhoid fever.

16. Acute appendicitis.

17. Melanotic sarcoma of scalp.

18. Subdural and subpial hemorrhage.

19. Hemorrhage into internal capsule and lateral ventricles.

20. Glioma of brain arising from medulla oblongata. Pressure on fourth ventricle; hydrocephalus.

21. Normal pregnant uterus.

22. Tuberculosis of lung, acute softening and caseation. Multiple bronchiectatic cavities.

23. Metastatic carcinoma of ribs, with multiple nodules beneath pleura. Primary epithelioma of neck.

24. Sarcoma of eye.

25. Hodgkin's disease. Cervical, axillary, and retroperitoneal lymph glands.

26. Diabetes mellitus. Atrophy and lipomatosis of pancreas.

27. Hypertrophic tuberculosis of mediastinal lymph glands extending along the pulmonary artery and bronchus. Death from hemorrhage into bronchus and trachea.

28. Incarceration of appendix in hernial sac. Multiple carcinomatous metastases of peritoneal surface of appendix. Carcinoma primary in gall-bladder.

Dr. D. J. McCarthy.

1. Thrombotic softening. Sylvian distribution.
2. Gelatinous (colloid) exudate in third cerebral ventricle.
3. Gelatinous exudate in third and fourth ventricles of brain.
4. Gelatinous exudate, with blood in the lateral ventricles.
5. Fibrosarcoma of temporosphenoidal lobe of brain.
6. Tumor of optic thalamus.
7. Brain. Showing results of trephinings for fracture. Remote results on cortex.
8. Tumor of optic thalamus.
9. Congenital absence of left optic nerve and absence of commissure.
10. Thrombosis of anterior cerebral artery.
11. Infiltrating glioma of spinal cord.
12. Cyst due to Bacillus capsulatus.
13. Sarcoma of temporosphenoidal lobe. General enlargement of right side of brain.
14. Embolic softening of medulla oblongata.
15. Hemorrhage into pons.
16. Subdural hemorrhage in a thirteen-weeks-old fetus.

Drs. C. K. Mills and **William G. Spiller.**

1. Destruction of Broca's area, causing motor aphasia.
2. Large sarcoma of brain removed by operation.
3. Sarcoma removed by operation from brain about three years ago. Patient still living.
4. Recent hemorrhage confined to optic thalamus.
5. Sclerosis of cortex in distribution of middle cerebral artery. The patient could say only "Anyone any."

6. Fibroma of pons.
7. External spinal pachymeningitis implicating the entire ventral surface of the spinal dura.

Drs. J. K. Mitchell and **William G. Spiller.**

Endothelioma of frontal lobe; operation. Death.

Dr. Charles P. Noble.

1. Appendix containing worm.
2. Fibromyoma.
3. Fibroma durum.
4. Abdominal pregnancy.
5. Fibroma uteri, with twisted pedicle. Dermoid cyst.

Dr. Charles C. Norris.

1. One-half ounce of a saturated solution of carmine injected into peritoneal cavity of a rabbit. Animal killed at the end of twenty-four hours. Slide shows a cross-section of the diaphragm; numerous carmine granules are seen in the lymph spaces.
2. Specimen shows the diaphragm of a rabbit injected intraperitoneally with one-half ounce of saturated solution of carmine granules, and killed at the end of twelve hours. The diaphragm is slightly studded with granules which are being eliminated from the peritoneal cavity through the organ.
3. One-half ounce saturated solution of carmine granules injected into the peritoneal cavity. Rabbit killed on second day. The section shows numerous fine granules in the lung in the process of elimination.

Dr. F. Savary Pearce.

1. Dural cyst.
2. Sarcoma, superior aspect of the cerebellum.
3. Tumor of hypophysis cerebri.
4. Sarcoma of infundibulum.
5. Hemorrhage into lateral ventricle.
6. Hemorrhage into internal capsule.
7. Sarcoma of pituitary body.
8. Extradural hemorrhage.

Dr. R. M. Pearce.

1. Normal pancreas of newborn child. Carmine injection.
2. Normal pancreas of newborn child. Island of Langerhans. Carmine injection.
3. Island of Langerhans in continuity with acinus. Pancreas of newborn child, stained with polychrome blue.
4. Syphilitic pancreatitis of newborn child.
5. Syphilitic pancreatitis of newborn child. Miliary gumma.
6. Syphilitic pancreatitis of newborn child. Islands of Langerhans, with ingrowth of connective tissue.
7. Syphilitic pancreatitis of newborn child. Mucoid degeneration of newly-formed connective tissue.
8. Pancreas of phosphorus poisoning. Osmic acid. Safranin and picric acid.
9. Hyperplasia of islands of Langerhans in cancer of pancreas.

Dr. G. E. Pfahler, 115 skiagraphs illustrating:

1. Diseases of the aorta (aneurysm, tortuosity, and dilatation).
2. Diseases of the brain (tumors, softening, and idiocy).
3. Diseases of the lungs (tuberculosis, hydropneumothorax, gangrene).
4. Diseases of the joints (tuberculosis, rheumatoid arthritis, the trophic change of locomotor ataxia, and "Chinese feet").
5. Atresia of the diaphragm, showing also the outline of the bowels, and congestion of the lungs.
6. Skiagraph, showing the shadow of the spinal cord.

Drs. George A. Piersol and **John M. Swan,** Museum of Medical Department, University of Pennsylvania.

1. Fracture of the skull by buckshot.
2. Tuberculous arthritis of hip.
3. Caries of tibia, with sequestrum.
4. Ankylosis of hip-joint.
5. Old trephine opening.
6. Fracture of skull.
7. Caries of femur.
8. Exostosis.
9. Rachitic lower extremity.

10. Osteosarcoma.
11. Vicious union after fracture.
12. Fracture of mandible.
13. Union after fracture of neck of femur.
14. Osteitis.
15. Osteomyelitis.
16. Osteoporosis.
17. Osteosarcoma, with pathologic fracture.
18. Osteomyelitis.
19. Superficial caries.
20. Vicious union after fracture (femur).
21. Septic osteitis.
22. Erosion of bodies of vertebra from pressure.
23. Exostosis (rider's bone).
24. Tuberculous caries of bodies of vertebræ.
25. Arthritis deformans affecting vertebræ.

Drs. **William Campbell Posey** and **E. A. Shumway.**

1. Tuberculosis of the iris.
2. Tubercle of the iris.
3. Tuberculous choroidoretinitis.

Drs. **C. S. Potts** and **William G. Spiller.**

Softening of pons, causing hemiplegia.

Drs. **W. E. Robertson** and **William G. Spiller.**

Cyst and glioma of vermis.

Dr. **J. F. Schamberg.**

Specimens of variolous skin preserved in Kaiserling's solution; prepared over a year ago.

Drs. **J. F. Schamberg** and **J. C. Wallis.**

Wax models of—

1. Eczema rubrum of hand.
2. Smallpox, eighth day.
3. Ulcerating syphiloderm of leg.
4. Nodular epithelioma of hand.
5. Evolution of vaccination. (Six models.)
6. Revaccinations. (Five models.)

*

Dr. George E. de Schweinitz.

1. Iridocyclitis and recurring hyphemia; detachment of retina. Eye had been quiescent for twenty-eight years, then cyclitis became active after a blow on the eye.

2. Leukosarcoma of choroid. No recurrence five years after enucleation.

3. Iridocyclitis caused by penetrating wound of lower ciliary region which caused sympathetic irritation. Early stages of membrane formation behind the lens well shown, as well as collection of round cells on surface of retina and within the perivascular lymph sheaths.

4. Melanotic sarcoma of choroid. Metastasis to the pleura, thigh bones, and probably the brain, and death within a year after enucleation.

5. Metastatic carcinoma of choroid. Patient died with symptoms of metastasis in the brain. No autopsy.

Drs. G. E. de Schweinitz and **E. A. Shumway.**

1. Obliteration of the filtration angle of the anterior chamber of the eye in glaucoma.

2. Excavation of optic nerve, glaucoma.

3. Absolute glaucoma following trauma; later recurring iridocyclitis.

4. Phthisis bulbi following slag burn of eye. From a patient in the Jefferson Medical College Hospital.

5. Phthisis bulbi following injury by powder explosion. From a patient in the Jefferson Medical College Hospital.

6. Traumatic iridocyclitis following blow on eye. From a patient in the Philadelphia Hospital.

7. Secondary glaucoma and staphyloma. Kettle-shaped cup in disk well shown.

8. Absolute glaucoma and bullous keratitis. From a patient in the University Hospital.

9. Absolute glaucoma and bullous keratitis; metallic foreign body imbedded in choroid for fourteen years.

10. Phthisis bulbi following corneal abscess. From a patient in the Philadelphia Hospital.

11. Phthisis bulbi. From Dr. W. F. Norris' collection in the University Hospital.

12. Phthisis bulbi. From a patient in the Philadelphia Hospital.

13. Total staphyloma of cornea. From Dr. W. F. Norris' collection in the University Hospital.

14. Total anterior staphyloma. From Dr. W. F. Norris' collection in the University Hospital.

15. Phthisis bulbi. From Dr. W. F. Norris' collection in the University Hospital.

16. Normal eye from a child one week old. From Dr. W. F. Norris' collection in the University Hospital.

17. Normal eye.

18. Normal eye. From a patient in the Jefferson Medical College Hospital.

19. Nontraumatic iridochoroiditis.

20. Adherent leukoma and atrophy of eyeball. From Dr. W. F. Norris' collection in the University Hospital.

21. Phthisis bulbi; detachment of retina. From Dr. W. F. Norris' collection in the University Hospital.

22. Iridocyclitis and atrophy of eyeball. From Dr. W. F. Norris' collection in the University Hospital.

23. Sarcoma of choroid. From Dr. W. F. Norris' collection in the University Hospital.

24. Sarcoma of choroid. From Dr. W. F. Norris' collection in the University Hospital.

25. Iridocyclitis from metallic foreign body which was removed by magnet. From a patient in the Jefferson Medical College Hospital.

26. Iridocyclitis with formation of cyclitic membrane. From Dr. W. F. Norris' collection in the University Hospital.

27. Sarcoma of the choroid. From Dr. W. F. Norris' collection in the University Hospital.

28. Sarcoma of choroid; large ciliary staphyloma. From Dr. W. F. Norris' collection.

29. Epibulbar sarcoma. From a patient in the Jefferson Medical College Hospital under the care of Dr. Sweet and Dr. de Schweinitz. The patient also had sarcoma at angle of jaw.

30. Iridocyclitis traumatica. From a patient in the Philadelphia Hospital.

31. Cyclitis of right eye following foreign body in vitreous (piece of copper). From a patient in the Jefferson Medical College

Hospital. Specimen shows in addition to cyclitis hemorrhages in anterior chamber and vitreous.

32. Iridocyclitis and detachment of retina. From Dr. W. F. Norris' collection in the University Hospital.

33. Epibulbar melanotic sarcoma. No recurrence one year later.

34. Glioma (neuro-epithelioma) of retina. From a patient in the Jefferson Medical College Hospital. Three years old; metastasis to the brain six months later.

35. Iridocyclitis following penetrating wound of eyeball. From Dr. W. F. Norris' collection in the University Hospital.

36. Sarcoma of Choroid. From Dr. W. F. Norris' collection in the University Hospital.

Dr. E. A. Shumway.

1. Normal angle of the anterior chamber of the eye.
2. Glioma of the retina.
3. Staphyloma of the cornea, following gonorrheal ophthalmia.
4. Iridocyclitis, following cataract extraction, in a highly myopic eye; posterior staphyloma, with choroiditis.
5. Anterior staphyloma following perforating ulcer.
6. Hemorrhagic glaucoma; rupture of globe.
7. Fetal eye, twenty-three weeks, showing hyaloid artery.
8. Phthisis bulbi, following burn of the cornea by nitrite of amyl.
9. Retained nerve sheaths in the retina.

Dr. Meyer Solis-Cohen.

1. Pachymeningitis interna hæmorrhagica. Patient exhibited symptoms of hemiplegia.
2. Acute arsenical poisoning; lung.
3. Acute arsenical poisoning; large intestines.
4. Acute arsenical poisoning; small intestines.
5. Acute arsenical poisoning; stomach.
6. Acute arsenical poisoning; esophagus.
7. Acute arsenical poisoning; heart, showing fragmentation of muscle fibers.
8. Acute arsenical poisoning; liver.
9. Acute arsenical poisoning; spleen.
10. Acute arsenical poisoning; testicle.
11. Acute arsenical poisoning; kidney.

Dr. William G. Spiller.

1. Sclerosis of first and second left temporal convolutions, causing word deafness.

2. Sarcoma of medulla oblongata and pons.

3. Old focus in optic thalamus, causing hemianesthesia and hemiplegia.

4. Tuberculous external pachymeningitis without deformity of the vertebræ.

5. Fracture of vertebræ, completely destroying the spinal cord at the seat of fracture.

6. Compression of brain from large hematoma (internal hemorrhagic pachymeningitis).

7. Endothelioma of frontal lobe; operation; death.

8. Brains from microcephalic idiots.

9. Large recent hemorrhage of brain.

10. Sclerosis of occipital lobe extending forward to the internal capsule, causing hemianopsia, hemiplegia, and hemianesthesia.

11. Internal hydrocephalus, causing symptoms of cerebellar tumor.

12. Primary sarcoma of spinal cord. The tumor entirely destroyed the cord in the thoracic region.

Dr. John M. Swan.

1. Secondary carcinoma of liver; from carcinoma of mammary gland.

2. Secondary carcinoma of lung; from carcinoma of mammary gland.

3. Frozen section of skull, showing result of pressure from a blood-clot.

Drs. W. S. Wadsworth and **William G. Spiller.**

1. Tumor of temporal lobe.

2. Hemorrhage of pons.

3. Hemorrhage in frontal lobe and occipital lobe from trauma.

Drs. Wm. M. Welch and **Jay F. Schamberg.**

1. Fetus born at term, showing eruption of smallpox in pustular stage; mother was suffering from smallpox.

2. Epidermal casts of the hand from a case of scarlet fever.

Dr. Courtland Y. White.

Tuberculosis of the liver and spleen of nylghai (deer) (Boselaphus tragocamelus). The chief lesions are in the liver and spleen. One mesenteric gland was the size of a man's fist, otherwise the peritoneal cavity was negative. Both lungs showed recent tuberculous lesions, but to a very slight extent.

Angiosarcoma of the Hypophysis Cerebri without Acromegaly.

JOSEPH McFARLAND, M.D.

The patient from whom the presented specimen was removed died in the Philadelphia Almshouse, and appears to have been but superficially studied during life, probably because her symptoms were too vague to suggest the importance of the lesion causing them.

She was first admitted to Blockley in 1884, and was subsequently admitted to the Almshouse on five different occasions. In 1891 she was admitted to the eye ward, where her eyes were studied and their condition pronounced incurable. Unfortunately, the clinical notes and treatment-sheets have been lost, and thus I was debarred from any knowledge that they might have thrown upon the case. The patient had no friends whose observation could be depended upon, so that it is not known whether her features had changed during the growth of the neoplasm or not. When the cadaver was examined no distinct signs of acromegaly could be detected. The hands and feet were of normal size and shape and lacked the usual characteristics of acromegaly. The history of the patient as gleaned from such notes as are preserved in the hospital contains but little of interest. After her eye affection was pronounced incurable she was transferred to the Almshouse again. She was nearly blind and partly demented, and gave very little information to those who questioned her.

For several trifling injuries to hips and shoulder, caused by falls, she was several times transferred to the surgical wards. It is said that she suffered from vertigo and from unsteadiness of gait, and frequently fell. After one of these falls she became very weak, was put to bed, and gradually sank into a condition of pronounced adynamia, and died. No autopsy was asked for at the hospital, and the interesting tumor was later discovered at the college after the body had been received there for teaching autopsy technic to the students.

Autopsy. The body was that of an aged white woman. There were no external marks or signs of disease. The examination of the body revealed very little of interest. There was an ordinary arteriosclerosis such as usually accompanies senility. The apex of the right lung contained a group of old latent or healed tubercles. The heart showed brown atrophy. The lungs were the seat of hypostatic congestion. The several organs were much atrophied. The kidneys showed chronic interstitial nephritis. Both pleural and peritoneal adhesions were present. They were old and rough, and their cause had long since disappeared.

The chief interest of the case centers about the brain. The brain itself was normal except for a marked sclerosis of its vessels.

The hypophysis cerebri, however, had apparently disappeared, and in its place was a large, flat, villous tumor that had so pressed upon the body of the sphenoid bone as to cause atrophy of all the clinoid processes. The tumor was irregularly rounded and flat, and was larger than a silver dollar. In its anterior growth it pressed upon the optic tracts and chiasm, which had undergone much atrophy and thus caused the patient's blindness. The tumor had no connection with the brain and did not invade either its substance, cavities, or membranes. In such parts of the growth as were subjected to microscopic examination no trace of the pituitary tissue could be detected, but as the specimen was to be preserved for the museum, the entire tumor was not cut up. The tumor was an angiosarcoma without peculiarities.

DISCUSSION.

DR. LLOYD said that he himself had had the pleasure of presenting a tumor of the pituitary body before this Society a few years ago. These tumors are rare, and are of interest in connection with the subject of acromegaly. In his case there had been no symptoms of acromegaly, and he was under the impression that very few tumors of the hypophysis that had been reported had been associated with such symptoms.

The specimen of cyst of the cerebellum shown by Dr. McFarland was one of the largest that he had ever seen. A number of such cases had been observed and studied by him, both during life and postmortem. His idea was that they were usually vascular in origin. The case reported by Dr. McFarland developed after scarlet fever. Such an origin is particularly likely in such an infection, and might be due to an embolus or thrombus. The same may be said of porencephalon, which is a cavity or cyst-like formation dating from an early period of life. While it is difficult to trace the origin of these cavities, they usually occupy the territory of supply of some particular artery, and are probably vascular in origin. Such a cyst of the cerebellum as shown by Dr. McFarland is, in fact, a form of porencephalon occurring in the cerebellum instead of the cerebrum.

Dr. Riesman said that several years ago in making autopsies at the Philadelphia Hospital he had encountered a number of cysts of the cerebellum. In these cases the condition was always vascular in origin. The cysts were much smaller than the one shown by Dr. McFarland. One case was of particular interest. There was a thrombotic obstruction of the posterior inferior cerebellar artery of one side, with a cystic development in the opposite cerebellar lobe. Upon examination the cause of this peculiar condition was found. There was only one posterior inferior cerebellar artery, and a branch of it supplied the opposite cerebellar hemisphere, the one that was the seat of the cystic change. That hemisphere, therefore, had a more precarious blood-supply to start with, and the vessel had a less rich anastomosis. The specimen is preserved in the museum, and Dr. Riesman hoped to exhibit it at some future time.

Dr. Steele recalled that Dr. Packard's classic case of acromegaly had shown sarcoma of the pituitary body.

Dr. Burr inquired as to the age of the patient with tumor of the pituitary body. He said that several years ago he had been dogmatic in stating that acromegaly is due to tumor of the pituitary body. After this he had found cases of tumor of the pituitary body in which there was no acromegaly; and, likewise, painful fatness and pituitary tumor, also without acromegaly. Nevertheless, tumor of the pituitary body is the most constant lesion, and the cases of tumor with absence of acromegaly generally occur in old persons.

Regarding the cyst, Dr. Burr thought that it was the result either of abscess or of thrombosis. He also remarked that a surgeon that diagnosed aneurysm on the strength of a bruit is more daring than any neurologist.

Dr. Coplin referred to Israel's exhaustive study of a case of acromegaly in which no lesion of the pituitary body was found. Recently, Cunningham has added his support to previously held opinions that there is a relation between acromegaly and giant-growth. It is held that when the morbid states giving rise to these conditions develop early they produce giant-growth. If the condition develops after the epiphyseal growth has ceased, it leads to acromegaly. There are, however, several instances on record that tend to controvert this view.

Dr. McFarland closed the discussion by saying that he regretted the absence of Drs. Laplace and Pearce, who were familiar with the clinical history of the case. He emphasized the fact that the lesions of the pituitary body are anatomic and physiologic. Hitherto, only the former have been studied, but the latter may be equally important. *November* 13, 1902.

A Fatal Case of Anthracosis, with Autopsy.

THOMAS STOTESBURY GITHENS, M.D.

The term anthracosis is used indifferently by medical writers for the deposition of carbon in the lungs and for the changes induced in the lung by such deposit. In the former sense it is a very common condition, and may be seen to some extent in every autopsy on old persons, especially in the city. It is, however, rare for the deposit to be of sufficient amount to cause structural change, and extremely so for it to be the direct cause of death. I was fortunate enough, while resident at Blockley, to see a case that proved fatal, and shall report it, with the kind permission of my chief, Dr. James Tyson. The history of the case is as follows:

Frank G., aged twenty-seven years, a barber, whose residence-place was Pottsville, entered the hospital on April 14th, complaining of pain in the chest. The family history is interesting, both parents having died of lung disease—the mother, of pneumonia; the father, of some obscure affection. The patient's previous history revealed the suggestive fact that he had had syphilis five years before.

He said that the present illness had lasted several years, but he could not set an exact date for its beginning. It started with dyspnea and cough, with slight pain in the side at times. These symptoms steadily increased; but he was able to work until the day before his admission, when he had a sharp, stabbing pain in the right side, accompanied by a chill. A severe hacking, exhausting cough started at the same time and continued until his death. On being questioned, he stated that he had had night sweats for the past year, but that there had never been any blood in the sputum.

Examination shows a young man of fair development, rather emaciated, sitting up and holding the sides of the bed to steady the accessory muscles of respiration. The respirations are 32 to the minute; and the pulse, 120, of quite high tension. Face anxious. Tongue heavily coated. Chest much emaciated; interspaces depressed; heart rapid, displaced, occupying the middle line. No murmurs.

Left Lung. Anteriorly: Vocal fremitus increased over the apex. Percussion note impaired from the second to the fourth rib. Auscultation shows, over this area, bronchovesicular breathing and fine, moist rales. Over the base of the lung the rales are much larger, and the breath sounds not so high-pitched. Posteriorly: Vocal fremitus increased over base. Percussion note generally impaired, but especially over scapula. Auscultation, same as anteriorly.

Right Lung. Anteriorly: Fremitus increased over base. Dulness and bronchovesicular breathing over base. Harsh breathing over middle of lung. Posteriorly: Similar, except for a small area of cavernous breathing and pectoriloquy in the suprascapular space.

Abdomen and *limbs* are negative.

The sputum examination was entirely negative. Tubercle bacilli were not found, and the amount of pigment was not sufficient to excite remark.

The urine was slightly albuminous and showed a few casts.

A diagnosis of tuberculosis was made in spite of the fact that the examination of the sputum was negative.

The patient's condition remained entirely unchanged until death occurred, in an attack of dyspnea, four days later. Toward the end he was somewhat delirious.

Autopsy. Body of a well-nourished adult male; rigor mortis absent. Pylorus 3 cm. below umbilicus. Liver 10 cm. below costal arch to sixth interspace. Pleural cavities totally obliterated. Pericardium normal. Heart displaced, occupying middle line. Spleen soft and pale. Adrenals normal. Kidneys: Capsule strips with difficulty. Pelvic fat increased. Cortex striated and injected.

Lungs. Pleura: There is a chronic plastic pleurisy, with marked thickening of the pleura and interlobar adhesions. Beneath the endothelial layer of the parietal pleura are sharply circumscribed areas of coal-dust infiltration, varying in size from that of a pin-head to half an inch long. There is considerable free pigment in the bronchi. Left lung: Has four lobes, one being merely a tab from the base. It is extremely heavy, and the entire lung sinks in water. Pieces of the upper and middle lobes sink, but those of the lower lobe float. On section, the lung is found to be intensely pigmented with coal-dust, and extensively fibrous. The middle and apical lobes are hard and rough, giving free pigment to knife and hands, and show no structure to the naked eye. Here and there are small areas of softening, with inky contents. The lower lobe shows irregular gray patches, varying in size from a point to an inch or more in length, and giving an appearance not unlike that of gray marble. No tubercles can be found. The peribronchial and mediastinal glands are large and black. Right lung: On section, appearances are much the same. In the posterior part of the upper lobe is a cavity, about the size of a walnut, filled with black fluid. There is no sign of tuberculosis.

Microscopic sections of these lungs show that the pigment granules are in part extracellular, in part enclosed in cells. The former form great masses around the bronchi in which no structure is visible, and where the lung is consolidated they are arranged in narrow bands, perhaps corresponding to the lymphatic vessels. Where the lung is not consolidated the pigment is always contained in cells of one of three types: (1) Far most numerous are large round cells, which correspond closely in size with the cells lining the alveoli, and may be either these or wandering connective tissue cells; (2) small round cells, like lymph corpuscles; (3) branched cells of the fixed connective tissue type. Any of these types of cells may be so filled that they constitute a solid black mass or they may contain only one or two granules. The flat cells lining the alveoli almost always contain pigment, and any granules lying free in the alveoli are enclosed in similar cells.

The lungs become solid through the formation of new connective tissue, which, by its contraction, cuts off the bronchioles and partly collapses the air cells. The epithelium lining these cells then desquamates, and the alveoli are filled by these cells and by wandering corpuscles. They are, at first, distinguished from the surrounding lung tissue by the absence of masses of pigment; but soon, by the deposition of pigment in them, the boundaries are lost, and it is no longer possible to determine the outline of pre-existing alveoli.

Of the special tissues, the pleura always contains pigment; and it is not uncommon to find deposits on the parietal pleura opposite masses on the visceral, where there are no adhesions. The cartilage of the bronchi rarely contains granules. I was unable to find any, but one writer has seen them in the cells. The lymph glands, of course, are always black; and microscopically we see the same sorts of pigment cells as in the lungs, in about the same proportion. A good deal of the pigment seems to be extracellular; many, if not all, of the original cells of the gland contain no pigment.

The shape and size of the individual pigment granules are very variable. A large number are long and narrow—too long to be carried in a cell. One of my sections shows a mass in the pleura as large as two or three cells.

Chemically, the pigment was about one-fifth of the lung by weight, and 6.23 per cent. of the pigment was ash. This proportion fitting hard coal best, we may assume that such was the pigment, and that the patient had worked in the mines before becoming a barber.

The first writers on anthracosis held that the carbon is endogenous. Mannkopf, Warburton-Begbie, Ritter, Koschlakoff, and even Virchow, all writing in the 60's, espoused this view. Arlidge, however, during the next decade, pointed out that in workers in steel, lime, etc., the pigment agrees chemically with the dust inhaled, and is not carbonaceous; and the experiments of Rosenthal, Slavjansky, Ruppert, Schottelius, and others, all done before 1880, and Greenhow's cases of pneumonokoniosis in stone-cutters, steel-grinders, and even flax-dressers, finally convinced the profession that the pigment is inhaled.

Kuborn, in 1862, and Mayet, in 1871, had held that the pigment is swallowed and deposited in the lungs by the lymphatics; but the experiments of Ruppert and Schottelius, before mentioned, in which rabbits were made to inhale powdered ultramarine, silenced this theory also. Having shown that the pigment is taken up by the lung itself, the question still remained as to the manner in which the pigment granules penetrate the tissues.

The first theory was that of Crocq, in 1862, who held that the cells lining the alveolar wall desquamate on account of the irritation of pigment particles, and that the granules force their way through the spaces thus left in the wall. The next view advanced was that of Traube, that the force of impingement of sharp particles drives them through the cells into the tissues. Several theories have since been advanced: Aufrecht's, that they force their way between the cells lining the bronchi, and that all the granules that reach the alveoli are expectorated; Rindfleisch's, that they are carried in by ameboid leukocytes; Bühl's, that the endothelial lining of the alveoli is related to lymphatic tissue, and is, therefore, absorptive; and Knauff's, that there are spaces between the cells of the alveoli through which they can pass. The last four are probably all true to some extent.

Whether sterile dust is, of itself, able to cause inflammation and consolidation of the lung, without infection, is a much more difficult question to decide. Most of the discussion has, however, been confined to the relation between tuberculosis and the lesions of anthracosis; and here we find the most opposing views. The strongest writer in favor of its tubercular nature is Tripier, who wrote in 1884, and again in 1894, reviewing all the literature and trying to show that all the reported cases were tuberculous. Most writers, however, consider that the dust is, of itself, sufficient to cause inflammatory change in the tissues. In any case, it is clinically distinct from tuberculosis, and the differential diagnosis is of importance. The most characteristic sign is the black sputum, but this is often absent. The next point, upon which I would lay special stress in the history of our case, is the extreme dyspnea, entirely out of proportion to the fever and weakness. Finally, the diffuseness of the process in the lungs and its long course, and in most cases the history of a dusty trade. The temperature in our case ranged from 104° to 99°; but, as the first observation was made only three days before death, it is impossible to draw conclusions from this. The respirations varied from 28 to 40, and the pulse from 100 to 140.

The points of extreme interest in our case are the youthfulness of the patient, the fact that his occupation was given as a barber, and the history of syphilitic infection five years before.

In conclusion, I should like to add a note in regard to the paucity of literature relating to anthracosis in this country. In a careful review of the subject, I have been able to find no mention of the disease in the United States, and only one from this continent—an article, published by Osler, in the *Canada Medical and Surgical Journal*, 1875. I wrote to the Secretaries of the County Medical Societies of the six counties of Pennsylvania in which most coal is mined, and they all answered that no cases or articles upon the subject had ever been presented before the societies. I wish to take occasion here to thank them for their prompt answers to my letters. *December* 18, 1902.

Atrophic Cirrhosis of the Liver.

SOLOMON SOLIS COHEN, M.D.,
AND
DAVID RIESMAN, M.D.

The specimens we show present nothing especially noteworthy, but, in their ensemble, afford such a good illustration of the morbid anatomy of cirrhosis of the liver that their exhibition in the fresh state seems warranted. They come from the body of M. L., a male Italian, aged forty-one years, admitted to the Philadelphia Hospital, October 6, 1902, with marked ascites. He had been a wine-drinker in Italy; but, after coming to this country, a year previously, he had formed the habit of drinking beer. Except for an attack of malaria nine years ago, he had always been healthy. There had been a sore on the genitalia fifteen years ago, but it had not been followed by an eruption.

Four months ago his abdomen began to enlarge, and he was twice tapped. Despite the ascites, his digestion was always good. Occasionally he had diarrhea, and at times precordial pain. On two occasions his nose bled rather profusely.

The patient was moderately emaciated. The conjunctivæ were slightly yellow. The abdomen was greatly distended with fluid, without any enlargement of the veins, except in the iliac regions. The liver was reduced in size, not reaching to the costal margin; the spleen was enlarged on percussion, but was not palpable; there were no hemorrhoids. The apex beat of the heart was in the third interspace; a murmur was not present. There was considerable edema of the legs. The urine varied in amount from 46 to 62 ounces; it had a specific gravity of 1012, and contained some albumin, but no sugar. Microscopically, red blood corpuscles were found. The blood-count was 4,560,000 erythrocytes and 7600 leukocytes. The differential count showed polymorphonuclear cells, 76 per cent.; eosinophiles, 4 per cent.; large lymphocytes, 13 per cent.; small lymphocytes, 7 per cent.

The patient was tapped a week after admission, and ten liters of straw-colored fluid were removed. The fluid had a specific gravity of 1010, and was rich in albumin; the sediment contained red blood cells and leukocytes. After tapping, the apex of the heart descended to the fifth interspace. The abdomen rapidly filled again. On October 16th the patient had a profuse nose-bleed. On October 21st, nine days after the previous tapping, he had to be tapped again, eight liters of fluid being withdrawn. After this the fluid accumulated more slowly. While the advisability of the Talma operation was under consideration, the patient became irrational and rather suddenly developed edema of the lungs, from which he died in the course of a few hours. During the days preceding death the urine had decreased to from five to eight ounces; but on the day before exitus it rose to twenty ounces. The patient had no fever at any time, except after the first tapping, when it rose to 101.4°, descending to 97° the next morning.

A very careful autopsy was made by Dr. McFarland, but it is unnecessary to give it in full. The patient's jaundice was distinctly noticeable at the necropsy. The abdomen contained between two and three gallons of straw-colored fluid. The liver was reduced in size, weighed 1360 grams, was yellowish in color, had a coarsely granular surface, and was of an enormously increased consistency. In every respect it was a typical cirrhotic liver. There were some adhesions between it and the diaphragm and other neighboring organs, but these were not very marked. The bile-ducts were patulous. The spleen was enlarged and weighed 750 grams. It was firm in consistency and showed distinct increase in the connective tissue. The intestines were pale in color. The veins of the omentum and of the mesentery were greatly engorged and very tortuous; and we have brought, preserved by Kaiserling's method, a piece of the mesentery that very beautifully shows this varicose condition of the vessels. The kidneys were somewhat enlarged—the left weighed 230 grams and the right 190 grams—but on section they showed nothing save slight parenchymatous degeneration. The lungs were edematous, and the left was densely adherent to the chest wall through the medium of very thick old adhesions.

REMARKS. The case is interesting from a clinical point of view for the following reasons:

1. The absence of the gastric symptoms that usually accompany cirrhosis of the liver. As we have repeatedly noted at the Philadelphia Hospital, gastrointestinal symptoms need not be present in otherwise typical cirrhosis of the liver.

2. The fact that nose-bleed was the only form of hemorrhage; ordinarily hemorrhages from the gastrointestinal canal predominate. The occurrence of epistaxis in cirrhosis of the liver is not easily explained on anatomic grounds, as there is no direct connection between the bloodvessels of the nose and the portal system. Bouchard[1] attributes it to ulcerative processes in the nose, and thinks it is more frequent from the right nostril than from the left.

3. The short duration of the disease—about five or six months.

4. The passage of large quantities of urine with increasing ascites. This was not due to a chronic interstitial nephritis, as the kidneys did not show such a condition at autopsy. It might be attributed to the use of diuretic remedies, had it not been present before these were administered.

5. The presence of considerable edema of the legs, which was not explained by any condition of the heart, unless the displacement of this organ had something to do with it.

November 13, 1902.

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[1] Bouchard: Revue de Médecine, October, 1902.

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